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Note: Performing your original search, ***"dopaminergic neurons" alzheimer***, in PubMed will retrieve *41* citations.

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1: J Neurochem. 1993 Sep;61(3):1024-34.

Immunocytochemical quantification of tyrosine hydroxylase at a cellular level in the mesencephalon of control subjects and patients with Parkinson's and Alzheimer's disease.

Kastner A, Hirsch EC, Herrero MT, Javoy-Agid F, Agid Y.

INSERM U289, Hopital de la Salpetriere, Paris, France.

Parkinson's disease is characterized by massive degeneration of the melanized dopaminergic neurons in the substantia nigra. The functional capacity of the surviving nigral neurons is affected, as indicated by the subnormal levels of tyrosine hydroxylase (TH) mRNA in these neurons and the presence in the parkinsonian mesencephalon of melanized neurons lacking TH immunoreactivity. This is apparently in contraction with the known overactivity of dopamine synthesis and release that occurs in the remaining dopaminergic terminals. To test the ability of the surviving neurons to express TH protein, a semiquantitative immunocytochemical method was developed. The relative amounts of TH were estimated with a computer-assisted image analysis system in the dopaminergic neurons of representative mesencephalic sections of control and parkinsonian brains and for comparison in brains from patients with Alzheimer's disease. In control brains, the mean TH content per neuron differed from one subject to another and between the different dopaminergic cell groups of the mesencephalon in the same subject. Within a given dopaminergic region, the level of TH was variable among neurons. In patients with Parkinson's disease, the ratio of TH protein content per neuron in the substantia nigra by reference to that of the central gray substance was reduced. In patients with Alzheimer's disease, the amount of TH was selectively reduced in the

remaining dopaminergic neurons of the ventral tegmental area, a region characterized by a loss in dopaminergic neurons. The decrease in cellular TH content might therefore be related to the presence of the neurodegenerative process in the area considered. In patients with Parkinson's disease, the incapacity of the surviving neurons to express normal TH levels may reduce the efficiency of the hyperactivity mechanisms that develop in the remaining striatal dopaminergic terminals.

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